



Vulnerability to stress: Personality facet of vulnerability is associated with cardiovascular adaptation to recurring stress

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ABSTRACT

It is increasingly suggested that personality traits are critical to understanding patterns of cardiovascular stress adaptation. However, studies have focused on higher-order traits with no research having examined underlying facet effects to repeated stress. The examination of facets provides a more granular examination, which has the potential to identify specific personality components that are relevant within the context of psychophysiological stress adaptation. This study objective was to determine if the underlying facets which encapsulate the dimension of emotional stability, are associated with cardiovascular adaptation to recurring stress. Continuous cardiovascular monitoring and psychometric measures were collated from 79 healthy young male and female adults, across a protocol of recurring active stress tasks. Multiple regression analysis revealed that the facet of vulnerability was associated with systolic and diastolic blood pressure adaptation across the protocol. More specifically, vulnerability was negatively associated with adaptation to recurring stress, such that those highest in vulnerability displayed a sensitization to the recurring stressor. No significant effects emerged for any other facet. Importantly, this research adds to the existing literature examining stress adaptation and has implications for future research on the relevance of examining facet effects. This study is the first to implicate the personality facet of vulnerability which encapsulates an individual's tendency to feel unable to cope with stress and becoming hopeless when faced with emergency situations, in the context of cardiovascular stress adaptation. Taken together, this study suggests that the facet of vulnerability is a critical component to consider in the context of cardiovascular stress adaptation.

1. Introduction

Possessing a biological basis and influenced by environmental factors (Yamagata et al., 2006), neuroticism is a higher-order personality trait which is a dimension that accounts for an individual's tendency to experience emotional instability. This broad trait has been linked to long-term mortality trajectories, including underlying cardiovascular mechanisms (O'Suilleabháin and Hughes, 2018). As with all higher-order traits, neuroticism is comprised of individual facets namely; anxiety, hostility, depression, self-consciousness, impulsiveness, and vulnerability. Facets of neuroticism have been linked to numerous health outcomes, which include the development of dementia in old age (Wilson et al., 2011) and vulnerability to coping with minor stressors (Felsten, 2004). Higher scorers on each facet can be described as follows: anxiety, the tendency to feel apprehensive, fearful, prone to

worry, nervous, tense, and jittery; hostility, the tendency to experience anger, and related states such as frustration and bitterness; depression, the tendency to experience guilt, sadness, hopelessness, and loneliness; self-consciousness, the tendency to be uncomfortable around others, sensitive to ridicule, and prone to feelings of inferiority; impulsiveness, the tendency to possess an inability to control urges and cravings; and vulnerability, the tendency to feel unable to cope with stress, becoming dependent, hopeless, or panicked when facing emergency situations (Costa and McCrae, 1992).

While much research has concluded that metabolically inappropriate cardiovascular reactivity (difference between stress response and baseline) can lead to an increased risk of cardiovascular health outcomes (e.g. Chida and Steptoe, 2010), it is increasingly being suggested that the observation that cardiovascular stress responsivity can demonstrate patterns of adaptation possesses significant health-

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relevant consequences (Hughes et al., 2018). As such, elevated cardiovascular responses to stress can be considered to possess positive outcomes if not maintained across time. For instance, short-term stress responsivity has been demonstrated to stimulate beneficial immune responding (Phillips et al., 2009). Importantly, personality have been observed as being associated with cardiovascular adaptation to repeated stress exposures (Hughes et al., 2011), across changes in active stress exposures (O'Suilleabháin et al., 2018a), across active and passive stress exposures (Soye and O'Suilleabháin, 2019), and during an acute stress exposure (O'Suilleabháin et al., 2018b).

Critical to developing an understanding of the association between personality and cardiovascular stress adaptation is the examination of facets. It has recently been reported that the facets may provide a much more nuanced approach to establishing the effects of personality on health-related processes (Soye and O'Suilleabháin, 2019). While each facet combine to provide an overarching higher-order trait dimension, it is quite clear that they represent very different components of emotional instability. In other words, the relevance of the facets will be dependent on the context they are expressed in, with each facet encapsulating a different component of emotional stability. The identification of specific aspects of emotional stability which are represented by facets, has the potential to provide avenues for future personalised intervention. For instance, in the context of being of relevance to cardiovascular adaptation to repeated stress, the facet of vulnerability would conceivably be of greater importance to the understanding of stress adaptation, than dimensions such as impulsiveness. While being the first to examine facet associations with cardiovascular adaptation to repeated stress, the present study sought to determine the predictive value of the facets which underpin the higher-order trait of neuroticism. Given the facet of vulnerability represents tendencies towards feeling an inability to cope with stress, it was felt this facet may provide the clearest associations with cardiovascular stress adaptation.

2. Method

2.1. Participants

This sample consisted of self-selecting healthy young adults ($N = 79$; $M \pm SD = 19.32 \pm 1.20$ years), comprising of both males ($N = 39$; $M \pm SD = 19.31 \pm 1.10$ years) and females ($N = 40$; $M \pm SD = 19.33 \pm 1.31$). Weight and height were digitally measured for all participants with body-mass index computed (BMI; $M \pm SD = 23.79 \pm 4.05$ kg/m²). Participants were free from cardiovascular related conditions, did not smoke, and displayed normotensive resting blood pressure measurements. Due to extensive research implicating blood pressure changes with age, the study was only made available to those between the ages of 18–25. A power computation demonstrates that the sample possesses > 95% power to detect large effects (Faul et al., 2007). Participants were voluntary, self-selecting, and recruited through an institutional sign-up system. Each participant received course credit for their participation.

2.2. Measures

2.2.1. Psychometric assessment

The complete NEO Personality Inventory (NEO-PI-R; Costa and McCrae, 1992) was administered. The higher-order factor of Neuroticism ($M \pm SD = 102.54 \pm 22.72$) and its underlying facets of Anxiety ($M \pm SD = 19.16 \pm 5.40$), Hostility ($M \pm SD = 14.94 \pm 4.95$), Depression ($M \pm SD = 17.70 \pm 4.41$), Self-Consciousness ($M \pm SD = 17.68 \pm 4.41$), Impulsiveness ($M \pm SD = 18.99 \pm 4.75$), and Vulnerability ($M \pm SD = 14.08 \pm 4.71$) were assessed within the present study (NEO-PI-R; Costa and McCrae, 1992). Examples of items are as follows; Anxiety (“I often worry about things that might go wrong”), Hostility (“I am known as hot-blooded and quick tempered”), Depression (“Sometimes I feel completely worthless”), Self-Consciousness (“When

people I know do foolish things, I get embarrassed for them”), Impulsiveness (“I have trouble resisting my cravings”), Vulnerability (“When I'm under a great deal of stress, sometimes I feel like I'm going to pieces”). Questions are scored on a Likert scale ranging from 0 (*strongly disagree*) to 4 (*strongly agree*). The NEO-PI-R has shown reliability, retest reliability, and validity through replication across languages, cultures, and ages (McCrae et al., 1998; McCrae and Costa Jr, 1997; McCrae et al., 2011; McCrae and Terracciano, 2005). Cronbach's α levels in the present sample are as follows (Anxiety = 0.80; Hostility = 0.76; Depression = 0.85; Self-Consciousness = 0.62; Impulsiveness = 0.70; Vulnerability = 0.75).

2.2.2. Physiological assessment

Continuous cardiovascular measurements were obtained using a Finometer (Finapres Medical Systems BV, BT Arnhem, the Netherlands), providing non-invasive beat-to-beat finger arterial measurements of blood pressure, HR, including various other hemodynamic parameters, such as, CO and TPR (Schutte et al., 2003). The Finometer meets the validation criteria of the Association for the Advancement of Medical Instrumentation, and has demonstrated to accurately measure cardiovascular function across multiple populations (Guelen et al., 2003; Kim et al., 2011; Schutte et al., 2003). Return-to-Flow Finometer calibration method was conducted for each participant.

2.2.3. Experimental task

A mental arithmetic task was employed as the stress task within the present study. The task consisted of a computerized series of subtraction problems, with participants required to enter correct responses under time pressure via a keyboard. The task employed the principle of standardized flexibility recommended for research cardiovascular stress responsivity (Hughes, 2001; Turner, 1994; Turner et al., 1986). This was achieved through subtraction items becoming more challenging or easier when three consecutive correct or incorrect responses were entered.

2.3. Procedure

Experimental protocol was conducted in a dedicated cardiovascular laboratory. Prior to attending for the study, all participants were instructed not to engage in any strenuous exercise or consume caffeine for 2 h prior to the commencement of their session. On arrival, participants were greeted by the researcher, provided with study information, with each providing written consent to partake in the study. Following this, participants' weight and height were digitally recorded. They were then sat in a comfortable chair with an arm support at a desk with a computer. While seated, the Finometer was attached to each participant's middle finger of their non-dominant hand. An acclimatization period of 20-min followed this, where participants were provided with none-motion reading materials (Jennings et al., 1992). Following this, the protocol comprised of; 10 min of a formal baseline period, 5 minute exposure to the stressor, 6 minute inter-task rest period, and a final 5 minute second exposure to the stressor. At the commencement of the experiment, each participant was informed of the order of phases, the duration of each phase, and what they would be required to do in each phase. The researcher was situated in the room throughout and sat behind an opaque screen.

2.4. Data reduction

Beat-to-beat cardiovascular measures of SBP, DBP, HR, CO, and TPR were recorded throughout the protocol using a Finometer. Data was then averaged into mean 1-minute readings, which were then averaged to create each phase (baseline period, exposure 1, rest, exposure 2). Firstly, reactivity for both stress exposures was calculated as the arithmetic difference between cardiovascular responses during the stress exposure and the baseline period. Following this, adaptation was calculated by subtracting reactivity during the second stress exposure from

Table 1
Means and Standard Deviations for cardiovascular values across experimental phases.

Variable	Baseline		Task 1		Inter-task rest		Task 2		Cubic effects	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>p</i>	Partial η^2
SBP (mm Hg)	120.36	8.57	130.97	12.83	126.58	10.72	129.91	12.50	< .001	0.392
DBP (mm Hg)	72.87	7.00	78.81	8.88	75.79	7.73	78.31	8.89	< .001	0.490
HR (bpm)	79.50	10.85	81.00	12.29	78.48	10.09	79.82	11.26	< .001	0.149
CO (lpm)	6.43	1.47	7.09	2.00	6.68	1.68	6.81	1.88	< .001	0.309
TPR (pru)	0.91	0.26	0.92	0.39	0.92	0.24	0.94	0.30	.52	0.005

reactivity during the first exposure. As such, values 0 to positive represented adaptation, whereas negative values signified lack of adaptation. This approach to examining adaptation has been used in existing literature (e.g. Lü et al., 2016).

2.5. Statistical analysis

In order to determine if the stress exposures successfully elicited cardiovascular stress responsivity, a series of repeated measures ANOVAs (SBP, DBP, HR, CO, TPR) were conducted. To assess cardiovascular adaptation, five hierarchical regressions were conducted with each facet of neuroticism entered as predictors while examining adaptation of each cardiovascular parameter. Each analysis also controlled for age, sex, and body mass index (BMI). These control variables were employed given they are well established as being associated with stress responsivity. Where a significant effect was observed for a facet, a supplementary analysis was conducted. This comprised of removing the non-significant facets from the model and examining the model comprising solely of the significant facet and covariates. This approach was taken to determine the degree to which the observed effects for the facet depended on the other facets in the model. Examinations of multicollinearity indicated a very low-level present within the study (see Table 2). When the assumption of sphericity was violated, Greenhouse-Geisser corrections were employed. Significant effects were graphically illustrated through the representation of facets as tertiles with trajectories across phases outlined.

3. Results

3.1. Elicitation of stress response

Significant main effects across protocol phases indicate that the stress exposures successfully elicited a cardiovascular stress response:

Table 2
Separate regression models for the facets predicting cardiovascular adaptation.

	VIF	SBP Adaptation			DBP Adaptation			HR Adaptation		
		B	β	<i>t</i>	B	β	<i>t</i>	B	β	<i>t</i>
Age	1.058	0.046	0.007	0.063	−0.004	−0.002	−0.15	0.019	0.005	0.044
Sex	1.445	−1.403	−0.090	−0.682	−0.695	−0.114	−0.891	−0.203	−0.021	−0.169
BMI	1.243	−0.274	−0.142	−0.142	−0.088	−0.116	−0.975	0.261	0.221	1.882
Anxiety	2.195	0.198	0.136	0.837	0.058	0.102	0.643	0.265	0.298	1.912
Hostility	1.440	−0.091	−0.058	−0.438	−0.065	−0.104	−0.816	−0.022	−0.022	−0.176
Depression	2.574	0.292	0.226	1.282	0.121	0.239	1.395	0.035	0.045	0.264
Self-Consciousness	1.709	−0.192	−0.108	−0.751	0.020	0.029	0.205	0.169	0.155	1.128
Impulsiveness	1.912	0.364	0.221	1.451	0.145	0.225	1.527	0.247	0.244	1.682
Vulnerability	2.427	−0.872	−0.524	−3.063**	−0.400	−0.616	−3.704***	−0.377	−0.370	−2.259*
		$(F(9, 69) = 1.54, p = .135, R^2 = 0.167, AdjR^2 = 0.058)$			$(F(9, 69) = 2.09, p = .042, R^2 = 0.215, AdjR^2 = 0.112)$			$(F(9, 56) = 2.39, p = .020, R^2 = 0.238, AdjR^2 = 0.139)$		

Note: Displayed values for R^2 and $AdjR^2$ represent the overall model including all facets and covariates.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

SBP, $F(2.38, 185.72) = 58.80, p < .001$, partial $\eta^2 = 0.430$; DBP, $F(1.973, 153.879) = 76.549, p < .001$, partial $\eta^2 = 0.495$; HR, $F(2.047, 159.704) = 6.189, p = .002$, partial $\eta^2 = 0.074$; CO, $F(1.893, 147.663) = 24.407, p < .001$, partial $\eta^2 = 0.238$; TPR, $F(1.424, 111.091) = 0.729, p = .441$, partial $\eta^2 = 0.009$ (see Table 1.).

3.2. Neuroticism

The higher-order trait of Neuroticism did not emerge as a significant predictor of cardiovascular reactivity or adaptation.

3.3. Cardiovascular reactivity and facets

For DBP, the facet of Vulnerability emerged as a significant predictor of reactivity to the initial stress exposure ($\beta = -0.352, p = .047$). The remaining facets were not significant. Following the removal of non-significant facets, the previously observed effect was no longer significant ($\beta = -0.098, p = .437$). No further significant effects were observed between facets and cardiovascular reactivity to both stress exposures.

3.4. Cardiovascular adaptation and facets

For SBP, the facet of Vulnerability was observed as a significant predictor of adaptation ($\beta = -0.524, p = .003$; see Table 2). No other significant effects were observed (all p 's $> .151$). Following the removal of non-significant facets from the model, the significant effect for Vulnerability remained ($\beta = -0.275, p = .028$), with a partial correlation of -0.252 . As such, higher levels of Vulnerability resulted in a sensitization of SBP to the recurring stress exposure. The model including Vulnerability and covariates was not significant $F(4, 74) = 1.826, p = .133, R^2 = 0.090, AdjR^2 = 0.041$. See full correlation matrix in Table 3.

Table 3
Correlation table of all variables.

	1	2	3	4	5	6	7	8	9	10	11	12
[1] Age												
[2] Sex	0.007											
[3] BMI	0.160	−0.017										
[4] Neuroticism	0.078	0.428**	0.086									
[5] Anxiety	0.112	0.394**	−0.129									
[6] Hostility	−0.001	0.204	0.040	0.779**								
[7] Depression	0.066	0.236*	0.162	0.839**	0.410**							
[8] Self Consciousness	0.038	0.270*	0.053	0.724**	0.545**	0.544**						
[9] Impulsiveness	0.084	0.411**	0.274	0.694**	0.347**	0.385**	0.336**					
[10] Vulnerability	0.041	0.427**	−0.016	0.789**	0.604**	0.450**	0.467**	0.468**				
[11] SBP Reactivity 1	0.124	−0.181	0.099	−0.024	−0.022	0.021	0.070	−0.039	−0.176			
[12] DBP Reactivity 1	0.111	−0.161	0.125	−0.015	−0.035	−0.043	0.106	−0.052	−0.146	0.889**		
[13] HR Reactivity 1	0.110	−0.009	0.321**	0.048	−0.027	−0.016	0.086	0.121	−0.091	0.427**	0.553**	
[14] CO Reactivity 1	0.110	−0.073	0.272*	0.021	−0.036	−0.025	0.049	0.149	−0.103	0.553**	−0.114	
[15] TPR Reactivity 1	0.090	0.038	−0.090	0.040	0.115	−0.058	0.082	−0.133	0.125	−0.266*	0.594**	
[16] SBP Reactivity 2	0.115	−0.040	0.148	0.092	0.050	0.061	0.168	0.185	−0.042	0.083	0.605**	
[17] DBP Reactivity 2	0.122	−0.043	0.151	0.090	0.041	0.007	0.164	0.141	−0.047	0.078	0.624**	0.798**
[18] HR Reactivity 2	0.060	−0.079	0.162	−0.152	−0.217	−0.164	−0.055	−0.031	−0.095	−0.115	0.244*	0.418**
[19] CO Reactivity 2	0.021	−0.155	0.267*	−0.028	−0.126	−0.033	0.025	0.053	0.040	−0.078	0.323**	0.337**
[20] TPR Reactivity 2	0.075	0.114	−0.126	0.016	0.036	−0.034	−0.004	−0.015	−0.020	0.112	0.138	0.228*
[21] SBP Adaptation	0.007	−0.155	−0.059	−0.131	−0.081	−0.046	−0.064	−0.133	0.005	−0.289**	0.418**	0.307**
[22] DBP Adaptation	0.001	−0.200	−0.020	−0.157	−0.119	−0.080	−0.075	−0.074	−0.017	−0.353**	0.531**	0.457**
[23] HR Adaptation	0.088	0.083	0.271*	0.255*	0.225	0.175	0.192	0.253*	0.292**	0.008	0.326**	0.336**
[24] CO Adaptation	0.146	0.117	0.037	0.076	0.133	0.009	0.042	0.020	0.180	−0.049	0.406**	0.319**
[25] TPR Adaptation	0.005	−0.052	0.028	0.013	0.045	−0.012	0.051	0.027	−0.067	0.002	−0.248*	−0.216
[1] Age												
[2] Sex												
[3] BMI												
[4] Neuroticism												
[5] Anxiety												
[6] Hostility												
[7] Depression												
[8] Self Consciousness												
[9] Impulsiveness												
[10] Vulnerability												
[11] SBP Reactivity 1	0.799**	−0.380**										
[12] DBP Reactivity 1	−0.202	0.387**	0.303**									
[13] HR Reactivity 1	0.417**	0.373**	0.241*	0.828**								
[14] CO Reactivity 1	0.538**	0.545**	0.033	0.279*	0.415**							
[15] TPR Reactivity 1	0.735**	0.796**	0.015	0.581**	0.408**	0.627**	−0.491**	0.444**				
[16] SBP Reactivity 2	0.691**	−0.165	−0.640**	−0.255*	0.107	0.066	−0.306**	0.213	0.874**			
[17] DBP Reactivity 2	−0.092	0.171	−0.286*	−0.471**	−0.253**	−0.047	−0.052	−0.214	0.054	0.138		
[18] HR Reactivity 2	−0.002	0.286*	−0.543**	−0.251*	0.071	0.071	0.245*	0.473**	0.737**	0.541**	0.438**	
[19] CO Reactivity 2	0.564**	0.501**	−0.254*	0.268*	0.279*	−0.146	−0.064	−0.821**	−0.670**	−0.462**	−0.013	−0.686**
[20] TPR Reactivity 2	0.247*	0.413**	−0.636**	−0.252*	−0.013	−0.063	0.328**					
[21] SBP Adaptation	−0.061	−0.119	0.783**	0.346**	0.074							
[22] DBP Adaptation												
[23] HR Adaptation												
[24] CO Adaptation												
[25] TPR Adaptation												

* $p < .05$.*** $p < .01$.

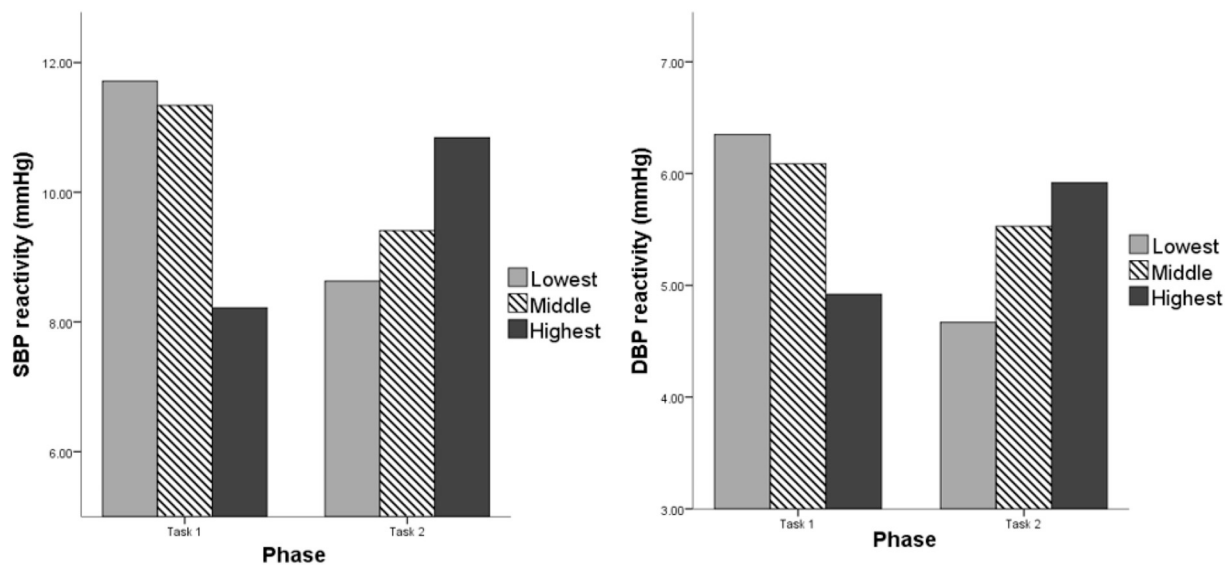


Fig. 1. Patterns of SBP, and DBP adaptation across both stress exposures by tertiles of Vulnerability. Note: tertiles are merely for illustrative purposes, analyses were conducted examining vulnerability as a continuous variable.

The facet of Vulnerability emerged as a significant predictor of DBP adaptation ($\beta = -0.616$, $p < .001$). No further significant effects were observed (all p 's $> .131$). Following the removal of non-significant facets from the model, the significant effect for Vulnerability remained ($\beta = -0.328$, $p = .008$), with a partial correlation of -0.302 . Similarly to SBP, higher levels of Vulnerability were associated with a sensitization of DBP to the recurring stress exposure (see Fig. 1). The model examining Vulnerability and covariates was significant $F(4, 74) = 2.723$, $p = .036$, $R^2 = 0.128$, $AdjR^2 = 0.081$.

For HR, the facet of Vulnerability was observed as a significant predictor of adaptation ($\beta = -0.370$, $p = .027$). No further significant effects were observed (all p 's $= .060$). Following the removal of non-significant facets from the model, the previously observed significant effect for Vulnerability did not remain ($\beta = -0.033$, $p = .790$), with a partial correlation of -0.031 . The model of Vulnerability and covariates was not significant $F(4, 74) = 1.691$, $p = .161$, $R^2 = 0.084$, $AdjR^2 = 0.034$.

For both CO and TPR, no significant effects emerged (all p 's $> .070$) Fig. 1.

4. Discussion

This study provides new evidence that the facet of vulnerability is predictive of a lack of cardiovascular adaptation to repeated stress exposures. More specifically, vulnerability was negatively associated with SBP and DBP adaptation to recurring stress. Following the removal of non-significant facets, a previous significant effect for HR adaptation was not observed. No other facets emerged as significant predictors of cardiovascular adaptation. Further, no significant effects emerged for both CO and TPR. The lack of effect observed within the present study for TPR is not without precedent, with active stress types which provide an opportunity for engagement known to elicit myocardial dominated stress responses. This study is the first to observe the facet of vulnerability as being of importance in the context of cardiovascular stress adaptation. In doing so, this study is also adding to existing literature demonstrating the importance of considering individual difference measures such as personality facets in the context of stress adaptation.

The findings observed in the present study are consistent with the theoretical conceptualisation of the facet of vulnerability. Vulnerability is a personality facet that accounts for the tendency for an individual to feel unable to cope with stress, and becoming dependent, hopeless, or panicked when facing emergency situations (Costa and McCrae, 1992).

In other words, this personality facet should theoretically be of critical importance to an individual's ability to adapt to recurring stress. Individuals higher in the facet of vulnerability should find repeated stress aversive, and the results observed within the present study supports this assertion. In doing so, this study is the first to suggest the relevance of a personality facet in the context of cardiovascular adaptation to repeated stress exposure. The examination of facets may provide a more granular and nuanced approach to the examination of individual difference effects on psychophysiological stress responses. This study also adds further evidence pertaining to the importance of the broader literature employing repeated stress exposures to provide a more complete representation of an individual's stress response profile. Importantly, these results suggest that Vulnerability may be solely relevant to stress adaptation, rather than stress reactivity. This has important implications for future research, particularly within the context of examination of stress across time.

While the present study possesses several strengths, which includes robust physiological and psychometric measurement tools, limitations must also be duly noted. While personality traits are considered to be relatively stable throughout the life course, it has been suggested that the trajectory of the development of personality in early adulthood continues until the age of approximately 30 (Costa and McCrae, 1997). As such, it is unclear how patterns of physiological responding observed in the present study translate across the lifespan. Research is required to determine the effects of facets in different laboratory stress contexts, whether with different acute stress exposures, passive stress, and during a stress exposure. Further examinations of facets within differing contexts will further elucidate the relevance of different facets in various contexts. Future research would benefit from incorporating multiple cardiovascular related biomarkers to provide a wider examination of the effects of vulnerability on health-relevant stress processes (O'Suilleabháin and Hughes, 2018). Further research is required to corroborate the present findings. While the present study did not use the inter-task rest period in the calculation of reactivity or adaptation, it is important to acknowledge that this period may also result in an artefact effect on adaptation. As such, future research should ideally employ a lengthy rest period to ensure each participant's cardiovascular responses return to baseline levels. The potential relevance of the facet of impulsivity within the context of stress reactivity requires particular attention given various self-report and behaviorally-assessed studies reporting its relevance to cardiovascular stress reactivity (Allen et al., 2009; Bennett et al., 2014; Diller et al., 2011). Similarly to Impulsivity,

the facet of Depression may be relevant to stress in a different context other than adaptation to repeated stress. The characteristics of the stress exposure in itself may drive the effect, such that differing facets are likely to be applicable in different stress contexts.

The present study builds upon and strengthens current literature on personality and cardiovascular stress adaptation. The findings suggest that the facet of vulnerability is of importance to cardiovascular adaptation to recurring stress. While being a significant addition to the existing literature on stress responding, this study is the first to examine the effects of personality facets on cardiovascular stress adaptation to repeated stress. The identification of individual facets provides a more granular examination of the effects of personality on stress responsivity, and provides a greater opportunity for research in the future refinement of potential stress monitoring and intervention. It is increasingly apparent that individual differences exist in the expression of psychophysiological stress processes, and that the suggestion that a single stress exposure is representative of an individual's pattern of stress responding is greatly limited. This research is building upon several important research avenues through the observations of vulnerability as significantly predicting cardiovascular adaptation to recurring stress.

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Declaration of Competing Interest

The authors declared no conflicts of interest.

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